

EXHIBIT 15

**SUR-REBUTTAL AND SUPPLEMENTAL EXPERT REPORT BY
DR. ALAN C. WHITEHOUSE**

A. Qualifications.

1. I am Dr. Alan C. Whitehouse. My address is 28810 North Milan Road, Chattaroy, WA 99003.

2. I am licensed in Washington and Montana. I am Board Certified in pulmonology and internal medicine. I currently practice chest medicine at the Center for Asbestos Related Disease (CARD) in Libby, Montana, where we have over 1,800 active cases of asbestos disease from exposure to Libby asbestos. I have practiced pulmonary medicine in Spokane, Washington, from 1969 - 2004, and in Libby, Montana, from 2004 to date.

3. My curriculum vitae is attached as Exhibit 1.

4. In addition, I have been an invited speaker on the subject of Libby asbestos disease at various locations across the country. See Exh. 1, Invited Presentations on Asbestos Disease.

5. Since 1980 I have evaluated or treated over 700 patients for asbestos disease from Libby asbestos. I have consulted with CARD physicians on many others. Since 1980 I have also evaluated or treated over 500 patients with asbestos disease from predominately chrysotile exposures. I am in a position to compare asbestos disease from Libby asbestos to asbestos disease from chrysotile asbestos. Chrysotile asbestos

is the usual form of asbestos used in building materials in the United States, accounting for about 95% of the total asbestos used in the United States. Fraser and Pare text, p.2420.

6. I have treated the entire range of pulmonary diseases. In my practice in Spokane in the years 1994-2004, the majority of my time, probably about 90%, was related to general chest diseases, including asthma, emphysema, lung cancer and the care of hospitalized patients. About 5-10% of my time was spent on asbestos related issues and other pneumoconioses. Probably about 10% of my time was related to industrial disease. Currently I spend a small amount of time on legal matters, but for the most part, my time is devoted to patient care and research.

7. In 30 years of practice I have probably testified at trial 10-15 times, about half for the plaintiff and half for the defendant. I testified in three asbestos trials relating to exposure from the W.R. Grace mine and mill near Libby, and one trial on the same subject in Missoula, Montana. These trials related to asbestos disease from Libby asbestos. In addition, my deposition has been taken on the subject of asbestos disease probably 25-30 times. I have testified in five Libby asbestos cases before the Montana Workers' Compensation Court.

8. I have published a paper on asbestos disease in Libby, titled

"Asbestos-Related Pleural Disease Due to Tremolite Associated with Progressive Loss of Lung Function: Serial Observations in 123 Miners, Family Members, and Residents of Libby, Montana," Am J Ind Med 46:219-225 (2004). A copy of the paper is attached as Exhibit 2. 123 patients were followed for an average of 35 months. Lung function was measured in terms of total lung capacity, forced vital capacity and diffusion capacity. The range of loss was 2-3% per year for each of these lung functions.

9. I have published a paper titled Whitehouse et al (2008), "Environmental Exposure to Libby Asbestos and Mesotheliomas." Eleven cases are discussed. A copy of the paper is attached as Exhibit 3.

10. Over the last three decades I have practiced occupational medicine. I have performed studies for companies, done screenings for companies and done disability exams for companies. In the 1980s I was involved in multiple asbestos disease screening programs for companies. I have also done independent medical examinations for the State of Washington Department of Labor and Industry for decades.

B. The mechanism for asbestos disease.

11. Asbestos is a mineral fiber. There are two kinds, serpentine (chrysotile) and asbestiform amphiboles. Chrysotile asbestos is the kind most often used commercially in building products. Chrysotile asbestos is

more curly, or more club-like, whereas amphibole asbestos is like tiny needles or spears. Libby asbestos is an amphibole. It has generally been referred to as tremolite. Using state of the art methodology, Meeker (2003), p.1959, has determined that the Libby asbestos is approximately 84% winchite, 11% richterite and 6% tremolite. Meeker, Figure 6 shows the incidence and close chemical form relation as between winchite, tremolite and richterite. Meeker, p.1967, suggests that:

The Vermiculite Mountain amphibole asbestos could, for the purposes of regulation only, be considered equivalent to tremolite or soda-tremolite asbestos in accordance with current and past industrial terminology for the Vermiculite Mountain amphiboles.

12. In relative terms of their length to width (aspect ratio), Libby asbestos fibers are long and sharp, like needles. The fibers breathed in are microscopic, as are the alveoli (tiny air sacs) in the lungs. When breathed in, the fibers lodge in the structure around the alveoli, and are too small to be expelled. Asbestos fibers irritate and inflame the lung tissue structure around the air sacs (the interstitia). Scarring in the interstitia is interstitial disease. When the interstitia are significantly scarred, they can no longer expand or contract fully, and breathing is restricted.

13. The amphibole fibers also migrate to the outside portion of the

lung, where they scar and inflame the pleura (the lung lining) and cause pleural disease. See the Frazer and Pare text, p.2809. Pleural disease seems particularly pronounced with Libby asbestos fibers.

14. The normal pleura is actually thinner than a blown up balloon. It is a very thin membrane, and it can expand like a balloon. Asbestos fiber scarring causes the pleura to look much like the orange portion of an orange rind, and can be just as thick. When surgeons peel it off the pleura, they call it a rind. When the lung lining becomes as thick as an orange rind, it can no longer expand freely and breathing is restricted. Asbestos disease is generally a restrictive lung disease.

C. Diagnosis of Asbestos-Related Disease.

15. For the diagnosis of asbestos related disease, we use the criteria of the American Thoracic Society (2004) Official Statement, "Diagnosis and Initial Management of Non-malignant Diseases Related to Asbestos," Am J Respir Crit Care Med, 170:691-715. Asbestos interstitial disease is due to scarring in the lung structure around the alveoli (air sacs) from the poking and inflammation from asbestos fibers. The asbestos pleural disease seen in Libby is due to the scarring and inflammation in the pleura (the lung lining) from asbestos fibers. The CARD Clinic generally uses "ARD" (asbestos related disease) to refer to non-malignant asbestos disease. Sometimes the

term "asbestosis" is used as an umbrella term, covering asbestos interstitial disease and asbestos pleural disease, since they are essentially the same disease process. The term would generally not be applied to a patient who has solely pleural plaques. The Rosenstock text, p.374, states: "Some investigators have used the term asbestosis to encompass non-malignant asbestos-related pleural abnormalities." Examples of investigators sometimes using "asbestosis" to encompass pleural abnormalities include the insulators studies. See e.g., Markowitz et al (1997), Table 2, where cases with pleural abnormalities and cases with ILO readings of 0/0 and 0/1 are included in "asbestosis" deaths. Horton et al (2006) publishes Libby ATSDR data. At Table 1 "asbestosis" ICD-9 501 is used to refer to "known asbestos-related diseases."

16. ATS Official Statement (2004) states the diagnostic criteria as follows:

Evidence of structural pathology consistent with asbestos-related disease as documented by imaging or histology.

Evidence of causation by asbestos as documented by the occupational and environmental history, markers of exposure (usually pleural plaques), recovery of asbestos bodies, or other means.

Exclusion of alternative plausible causes for the findings.

The ATS states that "the occupational history should emphasize

occupational and environmental opportunities for exposure that occurred about 15 years or more before presentation." Ruling out alternative causes requires a physical examination.

17. I have taken hundreds of work histories relating to asbestos exposure at the W.R. Grace mine and mill near Libby, Montana, and am familiar with conditions in the various jobs there. I have also taken hundreds of histories of exposure from family members of workers and Libby community members. Pathways for asbestos disease from Libby asbestos exposure are discussed at Peipins et al (2003). I have also evaluated hundreds of patients concerned about asbestos disease from Libby exposures, and have concluded that they do not have asbestos disease. For scores of these same patients, I have diagnosed lung disease other than asbestos disease.

18. Asbestos disease causes a restrictive defect. The amount of air breathed in is restricted. The physical examination includes determinations of chest restriction, the presence of rales (the crackling sound of scarred air sacs reopening), and an evaluation of shortness of breath. While chest x-rays occasionally show abnormalities not seen on CT scan, chest x-rays often miss parenchymal abnormalities of asbestosis seen on CT scan. See the Fraser and Pare text, p.2431. Chest x-rays miss an even higher

percentage of pleural abnormalities, as compared to CT scans. *Id.*, pp. 2431 and 2440. ATS Official Statement (2004), p.696, states: "Only 50 to 80% of cases of documented pleural thickening demonstrated by autopsy, conventional CT or high resolution CT (HRCT) are detected by chest radiograph (42, 43)." Frequently we see subpleural interstitial fibrosis on CT scans which is often not seen on chest x-ray, and which may play a significant role in the severity of the disease process. See the Schwarz and King text, p.422; ATS (2004) Official Statement, p.702.

19. At our clinic, lung function tests are performed in accordance with ATS criteria. We generally use Knudson norms for vital capacity (spirometry), Intermountain Thoracic Society for lung volumes, and Miller for diffusion capacity. Crapo norms are used as required for AMA Guides disability determinations. Abnormal values are under 80% of predicted or over 120% of predicted.

20. Pulmonary function tests are the measure of severity of asbestos disease. The Rosenstock text, p.370, states: "Pulmonary function tests are the most important tool for the functional assessment of non-malignant asbestos-related effects." Of all lung function tests, the three most important in asbestos disease are forced vital capacity (FVC), total lung capacity (TLC) and diffusion capacity (DLCO). The Fishman text, p.950, states "[t]he

characteristic pulmonary function changes of asbestosis are a restrictive impairment with a reduction in lung volumes (especially FVC and total lung capacity) decreased diffusion capacity, and arterial hypoxemia." ATS (2004) Official Statement, p.697 states: "Evaluation of subjects with suspected asbestos-related disease should include spirometry . . . all lung volumes and carbon monoxide diffusion capacity." The AMA Guides to the Evaluation of Permanent Impairment (5th Ed), p.107, "considers only pulmonary function measurements for an impairment rating."

For the Libby patients generally one percent predicted number under 60 (FVC, TLC, or DLCO) indicates severe lung function loss, one number 60-69 indicates moderate loss and one number 70-79 indicates mild loss. This is consistent with ATS (1991) Lung Function Testing, Table 13 values for FVC and TLC.

21. There are three components to pulmonary function tests. First is spirometry, which measures the amount of volume of the lung and the rapidity of inhalation, which gives an index of air flow and lung volumes. We usually do this before and after bronchodilator.

Second, we do lung volumes in what is called a body box, or plethysmograph, where we measure very small changes in air flow, pressure and volume, with a shutter and a closed system. Using Boyle's

law, one can calculate the volume of the lung.

Third, we measure diffusion capacity, by having the patient breathe a small percentage of carbon monoxide, using very tiny tracer amounts of methane, which is not absorbed, and we measure what comes out of the lungs. We measure the methane, measure the carbon monoxide, and the differential uptake gives us the carbon monoxide diffusion capacity. Diffusion capacity is the efficiency of the lungs in transferring oxygen into the blood stream. In restrictive lung disease there is interference with the air/blood interface due to the increased scarring that forms a barrier between the blood vessels and the alveoli (air sacs).

22. When asbestos disease due to Libby asbestos exposure is first diagnosable, there usually are no symptoms, only positive findings on chest x-ray or CT. The disease may take decades to progress to a point of severity. Severe disease may include shortness of breath, chest pain, rales, clubbing of the fingernails, hypoxia, cor pulmonale, pleural effusions, and oxygen dependency. See ATS (2004) Official Statement. At end stage the patient is bedridden, oxygen dependent, and generally the hypoxia will lead to organ malfunction and death.

D. Pleural Disease Generally.

23. "Pleural plaques" are a lesion of the parietal pleura, typically

presenting circumscribed borders and a "raised straight surface with clear cut edges when seen face on." ATS (2004) Official Statement, p.704.

24. The ATS (2004) Official Statement, p. 705, literature review collects many studies showing that pleural plaques are associated with decrements in lung function:

Studies of large cohorts have shown a significant reduction in lung function attributable to the plaques, averaging about 5% of FVC (forced vital capacity), even when interstitial fibrosis (asbestosis) is absent radiographically (74, 76, 107). The presence of circumscribed plaques can be associated with restrictive impairment and diminished diffusing capacity on pulmonary function testing, even in the absence of radiographic evidence of interstitial fibrosis (108, 109).

Textbook authors concur with the ATS (2004) Official Statement. *Fishman's Pulmonary Diseases and Disorders* (4th Ed 2008), p.945, states: "pleural disease has been recognized as the cause of reduced pulmonary function since the 1970s." The Fishman text, p.945, notes studies showing pleural plaques associated with decrements in FVC and FEV₁. *Fraser and Pare's Diagnosis of Diseases of the Chest* (4th Ed. 1999), p.2446, states: "it has become clear that asbestos related pleural disease can also affect lung function adversely (718, 721, 737, 738). Both pleural plaques and diffuse pleural thickening cause decreases in vital capacity, although the effects of diffuse thickening are more marked." Rosenstock et al, *Textbook of Clinical,*

Occupational and Environmental Medicine (2nd Ed. 2005), p.372, states:

"studies of asbestos exposed populations have variably observed small reductions in ventilation capacity associated with plaques."

25. Importantly, the ATS (2004) Official Statement, p.705, notes that in patients with pleural plaques, "decrements when they occur are probably related to early subclinical fibrosis." Schwartz (1990), p.321, concurs. Similarly, Whitehouse (2004), p.224, states: "pleural changes alone are unlikely to cause a decrease in DLCO (diffusion capacity). DLCO decreases are likely to be associated with interstitial disease not apparent clinically on either plain chest radiograph or HRCT." Pleural plaques are associated with losses of lung function, and are probably associated with subclinical fibrosis.

26. It is universally accepted that pleural plaques are markers of asbestos exposure. ATS (2004) Official Statement, p.705 states: "the presence of pleural plaques should be interpreted as a marker for elevated risk of malignancy." With or without pleural plaques, "it is clear that an excess risk for pulmonary carcinoma exists in asbestos-exposed workers in the absence of radiographic evidence of asbestosis." Fraser and Pare text, p.1075. The Rosenstock text, p.372, states: "plaques are also associated with increased risk for the malignant outcomes of asbestos exposure,

including mesothelioma and lung cancer." Edge (1979) established that an asbestos exposure sufficient to cause pleural plaques is sufficient to cause mesothelioma.

27. An overwhelming majority in the Libby cohort have not only pleural plaques, but also diffuse pleural thickening, a more serious form of pleural disease. Serial observations of Libby miners over 30 years show many with pleural plaques showing on chest x-rays in the 1960s or 1970s, then development of diffuse pleural thickening in the 1980s, then increasing lung function loss in the 1990s, with death in the 1990s or after 2000. See CD "plaques to interstitial disease 3/12/09" for two examples.

28. Diffuse pleural thickening is pleural fibrosis, which is scarring from an inflammatory process due to the presence of asbestos fibers. ATS (2004) Official Statement, p.707 states: "Diffuse thickening of the visceral pleura is not sharply demarcated, and is often associated with fibrous strands "crowsfeet" extending into the parenchyma (lung structure) . . . Diffuse pleural fibrosis extends continuously over a portion of the visceral pleura often causing adhesions to the parietal pleura." Page 707 also describes three kinds of diffuse pleural thickening by origin: (1) DPT "superimposed on circumscribed plaques," (2) DPT "after pleural effusion," and (3) DPT "caused by extension of interstitial fibrosis to the visceral pleura,

consistent with the pleural migration of asbestos fibers."

The Fishman text, p.946, states: "Diffuse pleural fibrosis occurs most commonly as part of a fibrotic process of the visceral pleura and subadjacent interstitium. It may occur, however, and be quite severe in patients with minimal pulmonary parenchymal fibrosis." This is true in the Libby cohort as well. Light and Lee, Textbook of Pleural Diseases, p.501, states: "Diffuse pleural thickening appears more closely related to amphibole than chrysotile exposure."

29. The ATS (2004) Official Statement, p.707, states: "Decrements associated with diffuse pleural thickening reflect pulmonary restriction as a result of adhesions of the parietal with the visceral pleura. Restrictive impairment is characteristic [meaning decreased vital capacity and decreased lung volumes]."

The Rosenstock text, p.373, states: "In contrast to the mild effect of plaques on lung function, diffuse pleural thickening may result in more significant restrictive respiratory impairment."

Schwartz (1990), p.1932, states: "Sheet metal worker (subjects) with diffuse pleural thickening had a lower forced vital capacity ($p < 0.001$), total lung capacity ($p < 0.01$), and CO diffusing capacity of the lung ($p < 0.05$) than those with normal pleura."